M-current imbalance induces a biphasic homeostatic plasticity, a fast relocation of the axon initial segment and a slow synaptic scaling

**ELSC cordially invites you to the lecture given by:**

**Prof. Bernard Attali**

Sackler Medical School in Tel Aviv University

**On the topic of:**

M-current imbalance induces a biphasic homeostatic plasticity, a fast relocation of the axon initial segment and a slow synaptic scaling

The lecture will be held on Thursday June 8th, 2017 at 17:00
Abstract:

Alterations in synaptic input, persisting for minutes to days, elicit homeostatic plastic changes. Here, we trigger a unique form of plasticity by targeting M-type K+ channels, which prominently localize to the axon initial segment (AIS) and regulate neuronal excitability. In hippocampal pyramidal neurons, acute M-channel blockage leads to neuronal hyperexcitability. However, minutes to hours of sustained M-current inhibition via cholinergic activation or direct channel block results in gradual reduction of intrinsic excitability. Dual soma?axon recordings combined with axonal Na+ imaging and immunocytochemistry reveals that these compensatory alterations are associated with fast a distal shift of the spike trigger zone and distal relocation of both Na+ and M-type K+ channels but not Ankyrin G. The rapid AIS homeostatic changes induced by M-current inhibition are contingent on the crucial AIS component, casein kinase 2. In contrast, prolonged (days) M-channel inhibition additionally leads to a slow synaptic scaling with decreased spontaneous firing and reduced mEPSC amplitude. Thus, M-channel imbalance results in a biphasic homeostatic plasticity process to stabilize network excitability, a fast AIS relocation, followed by a slow synaptic scaling.

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